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Adolescent Outcomes of Childhood Conduct Disorder Among Clinic-Referred Boys: Predictors of Improvement

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Much remains to be learned about the adolescent outcomes of clinic-referred boys whose childhood conduct problems are serious enough to meet diagnostic criteria for conduct disorder (CD). Six structured diagnostic assessments were conducted over 7 years of 73 clinic-referred 7–12-year-old boys who met criteria for CD in Wave 1. There were substantial individual differences in the adolescent outcomes of CD, ranging from worsening to sustained recovery, with most boys showing persistent, but fluctuating levels of CD. Improvement in CD was not accounted for by treatment or incarceration, but more positive outcomes over Waves 2–7 were predicted prospectively with substantial accuracy, using a combination of baseline predictors: less initial severity of CD, fewer symptoms of attention-deficit hyperactivity disorder, higher child verbal intelligence, greater family socioeconomic advantage, and not having antisocial biological parents.

KEY WORDS: conduct disorder; intelligence; socioeconomic status; antisocial personality disorder; longitudinal study.

Children are said to exhibit conduct disorder (CD) if they engage in multiple antisocial behaviors, including deception, physical aggression, and violation of property rights. Because CD is highly impairing to the individual and harmful to victims, it has been the subject of many studies. Although a great deal is known about the adolescent outcomes of childhood behavior problems in general (Farrington, Loeber, & Van Kammen, 1990; Fergusson, Horwood, & Lynskey, 1995; Nagin & Tremblay, 2001; Stattin & Magnusson, 1989; Verhulst & Van der Ende, 1992), less is known about the adolescent course of conduct problems in the subgroup of children whose problem behaviors are diverse and serious enough to meet diagnostic criteria for CD. Because it may be misleading to generalize findings on the longitudinal course of samples of children who mostly exhibit minor behavior problems to children who meet criteria for CD, it is important also to study the longitudinal course of the children at the extreme end of the behavior problem continuum who meet criteria for CD.

Some aspects of the longitudinal course of CD have been studied extensively (Frick & Loney, 1999). In a number of studies, children with serious conduct problems were reassessed in adulthood, with these studies suggesting that (a) adults who meet criteria for antisocial personality disorder (APD) almost always exhibited CD as children, and (b) although most children with CD show occupational and social dysfunction as adults, only about one third of children with CD later meet criteria for adult APD (Bardone, Moffitt, Caspi, & Dickson, 1996; Harrington, Fudge, Rutter, Pickles, & Hill, 1991; Robins, 1966, 1978; Robins, West, & Herjanic, 1975; Storm-Mathisen & Vaglum, 1994; Zoccolillo, Pickles, Quinton, & Rutter, 1992). What is not clear, however, is whether the children with CD who do not meet criteria for adult APD recover from CD during adolescence or continue to exhibit CD through adolescence, but do not meet the specific criteria for APD during adulthood.

Three longitudinal studies of population-based samples provide preliminary evidence on the adolescent course of conduct problems among children who met

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diagnostic criteria for CD in childhood. In the Isle of Wight Study, 35% of 93 children who met criteria for CD at 10–11 years of age continued to meet criteria for CD at 14–15 years of age (Graham & Rutter, 1973), but in a similar German study, most (8 of 11) boys who met criteria for CD at age 8 years met criteria for CD at age 13 years (Esser, Schmidt, & Woerner, 1990). In the Ontario Child Health Study, 45% of 36 children with CD when they were 4–12 years of age, met criteria for CD again at 8–16 years (Offord, Boyle, Racine, Fleming, et al., 1992). If the results of these three studies are combined to provide a tentative "meta-analytic" estimate of the stability of childhood CD into adolescence, only 56 of 140 (40%) children given the diagnosis of CD were given the same diagnosis again during adolescence. This suggests that slightly more than half of children with CD cease to meet criteria for CD sometime during late childhood or adolescence.

There are at least three reasons why additional research is needed on the adolescent outcomes of CD. First, in a preliminary report covering the first 4 years of the present study (Lahey et al., 1995), we found that boys who met diagnostic criteria for CD in the initial assessment showed levels of CD behaviors in subsequent waves that fluctuated above and below the diagnostic threshold for CD over time. As a result, estimating the persistence of CD from a single follow-up assessment greatly underestimates the stability of CD. It is possible, therefore, that these previous studies of the adolescent outcome of CD, which conducted only a single follow-up assessment, underestimated the persistence of CD. Second, Robins (1966) found a linear association between the number of childhood conduct problems and adult antisocial behavior among clinic-referred boys, suggesting that it is important to understand the course of CD at the level of the number of symptoms over time, rather than focusing only on the stability of the diagnosis. Third, the previous studies of the stability of children who received diagnoses used population-based samples and used diagnostic criteria for CD that were broader than the current *DSM-IV* definition. Although representative samples offer important advantages in such research, these studies did not demonstrate that the youths who received the diagnosis of CD were impaired enough to warrant clinical diagnosis and treatment. As a result of these issues, more data are needed to improve our understanding of the adolescent outcome of children with clinically-significant CD. Indeed, full knowledge of the adolescent outcome of childhood CD is currently the "missing link" in our understanding of the development of serious antisocial behavior from childhood to adulthood.

There is also a pressing need to identify childhood factors that predict which children with CD will have more or less favorable outcomes during adolescence. Identifying such early predictors will improve the prognosis of childhood CD and, if the predictors reflect modifiable causal processes that maintain CD over time, their identification may lead to improved methods of treatment. There is considerable published evidence from longitudinal studies of samples of children with conduct problems that were not limited to those who met diagnostic criteria for CD that may point to likely baseline predictors of the outcomes of childhood CD (Frick & Loney, 1999). Consistent evidence suggests that higher levels of conduct problems in childhood predict greater stability of future conduct problems (Loeber, 1982, 1991).

In addition, six studies of the outcomes of serious childhood conduct problems conducted in four countries found that intelligence is a significant inverse predictor of antisocial outcomes over 10 years or more (Farrington, 1991; Fergusson, Lynskey, & Horwood, 1996; Moffitt, 1990; Robins, 1966; Schonfeld, Shaffer, O'Connor, & Portnoy, 1988; Stattin & Magnusson, 1989). Because a recent longitudinal study of a nonreferred community sample did not find intelligence to be a significant predictor of the outcomes of aggressive children over time (Nagin & Tremblay, 2001) and because it is not yet clear whether verbal or nonverbal aspects of intelligence are more strongly related to conduct problems (Hogan, 1999; Lynam & Henry, 2001; Moffitt, 1993), this topic bears further study. In addition, there is emerging evidence that children with early onsets of CD, which tends to be more persistent over time than CD with later ages of onset, tend to exhibit comorbid attention-deficit hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD) during childhood (Lahey et al., 1998; Lynam, 1996; Moffitt, 1990). This suggests that childhood ADHD and ODD may predict the adolescent outcome of childhood CD.

In a follow-back study (Robins, 1966) and a retrospective study (Robins & Ratcliff, 1979), higher levels of antisocial behavior in the parents of boys with serious conduct problems predicted their adult antisocial behavior. Three previous longitudinal studies of nonreferred community samples also found that children with high levels of behavior problems tended to improve from childhood into adolescence if they were from higher socioeconomic status families and had mothers who did not give birth at an early age (Farrington, 1991; Nagin & Tremblay, 2001; Stattin & Trost, 2000). Robins (1966) and Robins and Ratcliff (1979), however, found that socioeconomic status accounted for little independent variance in predicting the adult outcomes of antisocial children after the children's initial levels of conduct problems were controlled.

Some theorists distinguish risk factors from protective factors for psychopathology (Masten, Garmezy,

Tellegen, Pellegrini, et al., 1988). Characteristics of children or families that are thought to be disadvantageous (e.g., low family income) are often conceptualized as risk factors, whereas positive characteristics (e.g., higher intelligence) are sometimes construed as protective factors. The designation of dichotomous predictors as risk or protective factors is arbitrary, however, and many predictors that are measured on continuous scales show linear associations with outcomes across their entire range. Stouthamer-Loeber et al. (1993) recommended conceptualizing continuous variables as protective or risk factors only when the theoretically adaptive or maladaptive tail of the continuous distribution is associated with better or poorer outcomes, but variation in scores across the rest of the distribution is unrelated to the child's outcome. Additional work is needed to evaluate the utility of this approach to continuous variables, such as intelligence and family income, which may predict outcomes of childhood CD.

Among social learning theorists, the focus has been on the role of parenting behavior in the outcomes of CD (Kazdin, 1985; Patterson, Reid, & Dishion, 1998). For example, it has long been known that there is a strong, inverse correlation between measures of parental monitoring and child and adolescent conduct problems in cross-sectional samples (Dishion & McMahon, 1998; Patterson & Stouthamer-Loeber, 1984). This correlation may not reflect a causal effect of parental supervision on child conduct problems for at least two reasons, however. First, cross-sectional studies cannot rule out the possibility of "child effects," that is, aversive interactions with children who engage in higher levels of problem behavior may lead some parents to "give up" and cease to monitor their children. Indeed, two longitudinal studies of children showed that higher levels of childhood behavior problems predicted greater reductions in parental monitoring over time, even when initial levels of monitoring were controlled (Kandel & Wu, 1995; Vuchinich, Bank, & Patterson, 1992).

Second, antisocial children tend to have antisocial and substance abusing biological parents (Eron & Huesmann, 1990; Lahey et al., 1988; Lahey, Russo, Walker, & Piacentini, 1989; Moss, Baron, Hardie, & Vanyukov, 2001), biological mothers who first gave birth at younger ages, and biological mothers who do not continue to live with the biological father (Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998). There is also evidence that parents with these characteristics are less likely than other parents to supervise their children closely (Loeber, Drinkwater, et al., 2000), but the causal links between these parental characteristics and child behavior problems may not involve monitoring. That is, lax parental monitoring could be a noncausal correlate of other parental characteristics that are causally linked in other ways to conduct problems in their offspring.

Child effects and the effects of parental characteristics (antisocial behavior and substance abuse) can be distinguished from influences of parental monitoring that are more likely to reflect causal effects, however, using longitudinal analyses that examine the association between levels of parental monitoring at Time 1 and later child antisocial behavior, while controlling both child antisocial behavior at Time 1 and background parental characteristics. One recent 2-year longitudinal study of low-income preschool children (Kilgore, Snyder, & Lentz, 2000) found that lax monitoring measured during the first assessment predicted child conduct problems in the second assessment, controlling for child conduct problems in the first assessment. This suggests that the correlation between parental monitoring and child behavior problems does not solely reflect child effects on parental monitoring, but parental characteristics were not controlled in this study to address the possibility that lax parental monitoring is a noncausal correlate of parental antisocial behavior, substance abuse, or other parental characteristics.

Social learning theorists have also hypothesized that parental use of physical punishment increases the likelihood of child behavior problems by encouraging maladaptive coercive cycles (Patterson et al., 1998). There is much cross-sectional correlational data in support of this hypothesis, but relatively few data from studies that allow stronger causal inferences. In support of the punishment hypothesis, however, two longitudinal studies of community samples have shown that maternal reports of greater use of physical punishment predicted subsequent increases in levels of late childhood and adolescent conduct problems when initial behavior problems were controlled (Cohen & Brook, 1995; Kandel & Wu, 1995).

Since 1987, we have conducted a longitudinal study of 177 clinic-referred boys who were 7–12 years of age at the time of their first assessment (Lahey et al., 1995). Data from this sample will be used to describe the course of CD into adolescence over the course of 6 years (from ages 7–12 years in Wave 1 through ages 13–18 years in Wave 7). Because the present study involved multiple reassessments of CD over time using contemporary criteria for CD, it provides needed information on the longitudinal course of CD among clinic-referred boys during late childhood and adolescence. Furthermore, the repeated assessments of the outcomes of CD allow tests of potential fixed (e.g., maternal age) and time-varying (e.g., parental monitoring) predictors of improvement in CD to determine if factors that predict outcomes in nonclinic samples generalize to a clinic sample of boys who meet

diagnostic criteria for CD. In these tests of predictors of outcomes, it is possible to control for the impact of potential confounds (such as juvenile detention or other residential placements that might reduce opportunities to engage in CD behaviors while detained) and to control for any influence of psychosocial or pharmacologic treatment on the course of CD. Finally, the present paper lays the foundation for a future report of the prospective relations between childhood CD and APD during adulthood when data collection in this study during early adulthood is completed.

METHOD

Participants

The participants were 73 boys who met diagnostic criteria for CD in Wave 1 from a longitudinal study among a sample of 177 boys who were outpatients at one of three mental health clinics when they were 7–12-years old. The larger sample of 177 boys was selected to be composed of approximately 75% boys with disruptive behavior disorders and 25% boys with other disorders (Loeber, Green, Lahey, Frick, & McBurnett, 2000). Of the 73 boys with CD in Wave 1, 69 (94.5%) were reported to have exhibited at least one symptom before the age of 10, suggesting that these boys had childhood-onset CD. Boys were ineligible for participation if they were mentally retarded, psychotic, taking medication that could not be discontinued for 2 days prior to their initial assessment, or their families were planning to move to another city. Eligible boys had to be living with at least one biological parent at the time of Wave 1, which was almost always the mother. Written informed consent was obtained from the parent or legal guardian and oral assent or written consent was obtained from the boys in each wave. Participants were reassessed during Years 2, 3, 4, 6, and 7, with no assessment wave during Year 5 due to budgetary limitations.

Assessment Measures

The Wechsler Intelligence Scale for Children—Revised (Wechsler, 1974) was administered during Wave 1. In each assessment wave, the boys and parents were interviewed separately using the NIMH Diagnostic Interview Schedule for Children (DISC; Costello, Edelbrock, Kalas, & Dulcan, 1984), which queried *DSM-III* and *DSM-III-R* symptoms of ADHD, CD, ODD, overanxious disorder, separation anxiety disorder, dysthymia, and major depression occurring during the last 6 months. The DISC has acceptable test-retest reliability (Edelbrock et al., 1985), discriminates clinic-referred youth from those without need of treatment (Costello, Edelbrock, & Costello, 1985) and correlates substantially with standardized parent ratings (Edelbrock & Costello, 1988).

Parent and youth reports of symptoms from the DISC were used consistent with previous findings on the relative reliability and validity of parent and youth reports of various types of symptoms (Hart, Lahey, Loeber, & Hanson, 1994; Jensen et al., 1999; Loeber & Lahey, 1989). The boys' numbers of symptoms of ADHD and ODD were assessed by summing the number of the *DSM-III-R* symptoms of each disorder reported by the parent. The DISC queried both informants about *DSM-III-R* symptoms of CD and the *DSM-IV* symptom of bullying. In addition, the parent was asked about the *DSM-IV* symptom of staying out late without permission. Thus, it was possible to write an algorithm for the diagnosis of *DSM-IV* CD in Wave 1, except that the *DSM-III-R* symptom of frequent lying was used instead of the more restrictive *DSM-IV* symptom of lying to con others. The "or rule" was used to combine reports of CD behaviors, with behaviors considered to be present if reported by either the parent or the youth (Piacentini, Cohen, & Cohen, 1992). Depression was similarly measured by summing the number of nonoverlapping *DSM-III-R* symptoms of major depression and dysthymia reported by the parent or the youth. Anxiety was assessed by summing the number of *DSM-III-R* symptoms of overanxious disorder and separation anxiety disorder reported by either the parent or the youth.

The parent was also interviewed in Wave 1 to assess *DSM-III-R* mental disorders of the child's biological mother and father using the sections on APD and substance abuse from the Schedule for Affective Disorders and Schizophrenia (SADS; Spitzer & Endicott, 1967) and the section on mood disorders from the Structured Clinical Interview for *DSM-III-R* (SCID; Spitzer, Williams, & Gibbon, 1987). Caspi et al. (2001) found that mothers' and fathers' reports of fathers' antisocial behavior agreed at a high level, but that maternal reports were conservative, as mothers underestimated fathers' antisocial behavior somewhat. Pfiffner, McBurnett, and Rathouz (2001) similarly provided evidence supporting the validity of maternal reports of the antisocial behavior of fathers using family-study methods. The SADS and SCID interviews covered varying time periods (lifetime for substance abuse and APD and past 12 months for depression). In a different sample, Lahey et al. (1989) found that APD was uncommon among the biological mothers of boys with CD, but a well-validated index of antisocial personality created by taking the mean of the K-corrected F, Psychopathic Deviate, and Mania scales of the Minnesota Multiphasic

Table I. Descriptive Statistics for Potential Baseline (Wave 1) Predictors of Individual Differences in the Outcomes of CD (During Waves 2–7) Among 73 Boys Who Met Criteria for CD in Wave 1

Demographic and socioeconomic factors assessed in Wave 1	
Race–ethnicity	64.4% non-Hispanic White; 35.6% African American
Biological parents status	30.1% married or cohabiting partners in Wave 1
Total family annual income	Mean = 21,219; *SD* = 18,755
Maternal education in years	Mean = 12.3; *SD* = 2.57
Maternal age at first birth	Mean = 19.7; *SD* = 3.16
Child characteristics assessed in Wave 1	
WISC-R verbal intelligence	Mean = 98.2; *SD* = 15.5
WISC-R performance intelligence	Mean = 97.6; *SD* = 16.1
Number of CD symptoms	Mean = 4.3; *SD* = 1.2 during last 6 months
Number of ADHD symptoms	Mean = 6.4; *SD* = 4.0 during last 6 months
Number of ODD symptoms	Mean = 6.7; *SD* = 1.8 during last 6 months
Number of anxiety symptoms	Mean = 5.0; *SD* = 3.1 during last 6 months
Number of depression symptoms	Mean = 2.5; *SD* = 2.3 during last 6 months
Maternal psychopathology and substance abuse assessed in Wave 1	
MMPI antisocial index	38.0% *T*-score >60
Substance abuse	8.2% lifetime
Major depression	19.2% last 12 months
Paternal psychopathology and substance abuse assessed in Wave 1	
Antisocial personality disorder	31.5% lifetime
Substance abuse	48.0% lifetime
Major depression	12.3% last 12 months

Note. WISC-R = Wechslser Intelligence Scale for Children—Revised; ADHD = attention-deficit hyperactivity disorder; ODD = oppositional defiant disorder; MMPI = Minnesota Multiphasic Personality Inventory.

Personality Inventory (MMPI; Huesmann, Lefkowitz, & Eron, 1978) was strongly related to CD in their sons. Following standard practice for MMPI *T*-scores, this index was dichotomized for the present analyses rather than treating it as a continuous variable, with *T* scores of $\geq 60 = 1$ (which corresponds to 1 *SD* above the mean of the MMPI standardization sample) and *T*-scores $<60 = 0$ (Hathaway & Monachesi, 1952). Descriptive statistics for these potential baseline predictors are presented in Table I.

In each wave, the parent was asked if the child had taken any type of prescribed psychoactive medication for problems of attention, emotion, or behavior for any length of time or had received one or more sessions of psychotherapy in the past 12 months. In order to determine if juvenile detentions or inpatient hospitalizations influenced levels of CD behaviors due to lack of opportunity to engage in them, the parent was asked if the boys experienced each type of such confinement for one night or longer in each wave. Also, the parent was asked 14 questions about parental monitoring of the boy during the last year in each wave, using a version of a measure that has adequate internal consistency and has been found to be correlated with delinquent behavior in a community sample of boys (Loeber et al., 1998). These items queried the extent to which the boy was under direct adult supervision, the parent's knowledge of the boy's location and companions when away from home, the boy's knowledge of the parent's location when away from home, the parent's setting and implementing of curfews for the boy, and the parent's discussion of the boy's plans for the coming day and events of the current day. These items were scored 0, 1, or 2 and summed, with higher scores indicating more parental monitoring. In the full sample of the Developmental Trends Study ($n = 169$) with complete data over waves, Cronbach's alpha for the monitoring measure increased steadily from $\alpha = .50$ in Wave 1 to $\alpha = .76$ in Wave 7. The lower internal consistency coefficients in the earlier waves appear to reflect a very narrow range of parental monitoring scores in the early waves that increased as the boys grew older. The parent was also asked one item in each wave about corporal punishment (slapping, spanking, or hitting the boy with an object), which also has been found to be correlated with delinquency in a community sample of boys (Loeber et al., 1998). Responses were scored 0, 1, or 2 and summed, with higher scores indicating more corporal punishment.

Data Analysis

Among the 74 boys who met criteria for CD in Wave 1, one boy was not interviewed in Waves 4, 6, and 7 and four boys could not be interviewed in one wave each (Wave 3 in two cases and Wave 6 in two cases). Our data analytic approach can estimate functions for subjects

with missing data, but the one subject with missing data in all of the last three waves was dropped from all analyses, leaving a sample size of 73. The outcome variable in all analyses was the number of CD behaviors in each of five follow-up waves after baseline (Waves 2–7, excluding Wave 5). These were treated as count variables (integers reflecting the continuous number of CD behaviors in each wave from 0 to 11). As is typical of symptom counts, each distribution was highly skewed, with modal values being at or near 0, with few boys exhibiting high numbers of symptoms. The proportion of boys with 0 or 1 symptoms of CD in each of Waves 2, 3, 4, 6, and 7 was 35.6, 29.6, 31.5, 31.0, and 35.6%, respectively. Such data do not meet the assumption of normality required by most classical approaches to longitudinal data analysis, such as repeated-measures analysis of variance. One appropriate approach to such skewed count data is to model the mean numbers of behaviors in longitudinal log-linear regression models implemented in generalized estimating equations (GEE; Zeger & Liang, 1986). GEE models the average value of the outcome variable for each subset of individuals who share the same value of the predictor variable. Because GEE estimates averages, and not the entire distribution of values, it is less restricted by distributional assumptions than other approaches to longitudinal data analysis. GEE also allows the user to specify a within-person correlation structure to account for within-person correlations in the outcome variable over time. In the present analyses, an unstructured correlation matrix was specified in all GEE analyses (except where noted for time-lagged models) and the Poisson distribution was used as the working model for the counts of CD behaviors. The distributions of symptom counts were "overdispersed" relative to the Poisson distribution, as the variance exceeded the mean in each wave. However, all statistical tests in the present GEE analyses were based on the robust (*empirical*) standard error because it automatically adjusts for overdispersion and reduces concern about correct specification of the within-person covariance structure. Thus, GEE allows unambiguous interpretation of analyses of counts of symptoms when the distributions take the shape of the present data, which means that it is an appropriate option for many similar studies in developmental psychopathology.

Potential baseline predictors of CD outcomes over Waves 2–7 were measured in Wave 1 and treated as time-fixed covariates. In these GEE models, the regression coefficients (β) have the interpretation of the log relative mean number of CD behaviors over Waves 2–7 associated with a one-unit difference in the predictor. For example, in the left-hand column of Table II, the estimate of $\beta = .33$ for the dichotomous variable of maternal antisocial personality means that the mean number of CD symptoms is estimated to be 39%, $\exp(.33) = 1.39$, greater during Waves 2–7 among boys with an antisocial biological mother than among boys without an antisocial mother. Interpreting β as an estimate of effect size in this way requires a recognition that although some baseline predictors are dichotomous, the range of scores for other predictors (waves, age, maternal age at first birth) are more continuous. Therefore, a smaller β coefficient for a predictor with many units might indicate a stronger effect than a larger β for a predictor with fewer units.

Similar GEE log-linear models were used to assess the association of the time-varying covariates of treatment and incarceration with the level of CD behaviors in the same waves. Other log-linear models with time-varying covariates were used to assess the role of parenting in the outcome of CD. Here, parenting variables in each wave (Wave t) were included as covariates in models where the response is the number of CD behaviors in the following wave (Wave $t + 1$). In these models, the temporal association of parenting and CD was adjusted for the number of CD behaviors in the preceding wave by including CD at Wave t as another time-varying covariate. These models were fitted using GEE with an independent correlation structure. All tests of significance for predictor variables in the GEE analyses were based on the z-statistic and all used 1 *df*.

RESULTS

Longitudinal Course of Childhood CD

We first examined possible developmental trends in CD behaviors over time (see Fig. 1) to provide a framework for further analyses. In the first longitudinal log-linear analysis in GEE, time was treated as a continuous variable, with values of 1, 2, 3, 4, 6, and 7, corresponding to the six assessment waves conducted over the 7-year period. The boys' age at the time of the first assessment was included in these models to assess possible differences in the course of CD over time among boys with different ages at the start of the study. In this joint model, the quadratic term for time (waves) was significant, $\beta = .01, z = 2.68, p < .01$, as was the boys' age at entry into the study, $\beta = -.07, z = -2.63, p < .01$. Two subsequent longitudinal analyses were conducted to understand the nonlinear changes in CD over time reflected by the significant quadratic effect for time. First, when the number of CD behaviors during only Waves 1 and 2 were treated as the response variables, there was

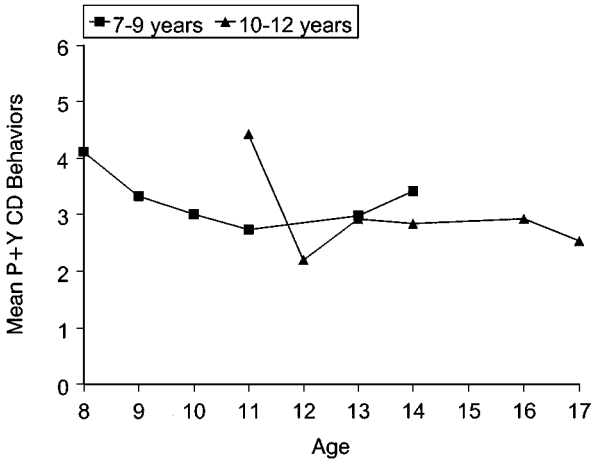

Fig. 1. The mean number of conduct disorder behaviors reported by the parent and youth (P + Y) across Waves 1–7 for boys who were 7–9 years of age (average of 8 years) or 10–12 years of age (average of 11 years) in Wave 1.

a marked decline in the number of CD behaviors from Wave 1 to Wave 2, $\beta = -.43$, $z = -4.91$, $p < .0001$, but the boys' age at the start of the study was not associated with the number of CD behaviors during Waves 1 and 2, $\beta = -.03$, $z = -1.01$, $p = .31$. When the age-by-time interaction was added to this model, however, it revealed that the decline in CD behaviors from Wave 1 to Wave 2 was greater for older boys than for younger boys, $\beta = -.14$, $z = -2.78$, $p < .01$.

In the second analysis conducted to understand the nonlinear decline in CD over time, the number of CD behaviors during Waves 2, 3, 4, 6, and 7 (the period of the assessment of the outcomes of CD after Wave 1 in the present study) were treated as the response variables. In this model, there was not a significant effect for time (waves), $\beta = .01$, $z = 0.37$, $p = .71$, controlling for differences associated with the boys' age at the start of the study, $\beta = -.09$, $z = -2.71$, $p < .01$. When the age-by-time interaction was added to this model, it was not significant, $\beta = -.01$, $z = -0.81$, $p = .42$. Therefore, as shown in Fig. 1, these initial longitudinal analyses reveal that there was a marked decline in the mean number of CD behaviors from the start of the study to the next annual assessment (Waves 1–2), particularly among older boys. The decline in CD behaviors from Wave 1 to Wave 2 may partly reflect regression to the mean following the youth's referral to a clinic during a time of peak symptoms. During the period in which the outcomes of CD were assessed prospectively (Waves 2–7), however, there was no significant change over waves in the average number of CD behaviors.

Thus, there were two indications of possible developmental trends that must be taken into consideration in describing the adolescent outcomes of childhood CD. The significant term for the boys' age at the start of the study in all models reflects slightly, but consistently lower mean numbers of CD behaviors across all waves among boys who were older at the time of entry into the study. In addition, the decline in CD behaviors from Wave 1 to Wave 2 was greater in older than younger boys. Because this unexplained age-by-time interaction was limited to the change in CD behaviors from Wave 1 to Wave 2, it does not influence the evaluation of outcomes of CD after Wave 1. On the other hand, the finding that boys who were older in Wave 1 had lower numbers of CD behaviors during the period in which we are assessing the outcomes of CD (across Waves 2–7) could indicate one of two things. First, there may be a gradual decline in the number of CD behaviors with increasing age among boys who are referred to clinics during elementary school (see Fig. 1). Second, selection effects may have operated in this sample, such that boys who entered the study at earlier ages might have had more serious and persistent CD. In either case, it is necessary to control the boys' age at the start of the study in all subsequent analyses of the outcomes of CD after Wave 1 and analyses of the variables that predict individual differences in these outcomes.

It is important to note that the lack of a significant effect of time on the *average* number of CD behaviors during Waves 2–7 masks a marked degree of heterogeneity in the outcomes of individual boys during this 5-year period. The wide range of individual differences in outcomes of CD is illustrated in Fig. 2 by plotting the number of CD behaviors of each boy in each assessment wave. In order to facilitate interpretation of these 73 single-subject plots, the boys who met criteria for CD in Wave 1 were arbitrarily divided into three *illustrative* groups based on their average numbers of CD behaviors during Waves 4–7. The 17 boys in the left panel of Fig. 2 exhibited a mean of $\geq$4.0 CD behaviors during Waves 4–7. Although they showed fluctuations in the number of symptoms over waves (above and below the diagnostic threshold of three symptoms for CD), no boy in this panel exhibited $<$1.0 CD behavior in any wave. The 47 boys in the middle panel exhibited a mean of 1.0–3.9 CD behaviors during Waves 4–7. These boys also showed marked fluctuations in their numbers of CD behaviors over time; nearly one-third exhibited no CD behaviors during at least one of Waves 4–7, but all continued to exhibit significant levels of CD behavior over time. Only the 9 boys in the right panel (mean number of CD behaviors in Waves 4–7 of $<$1.0) were in

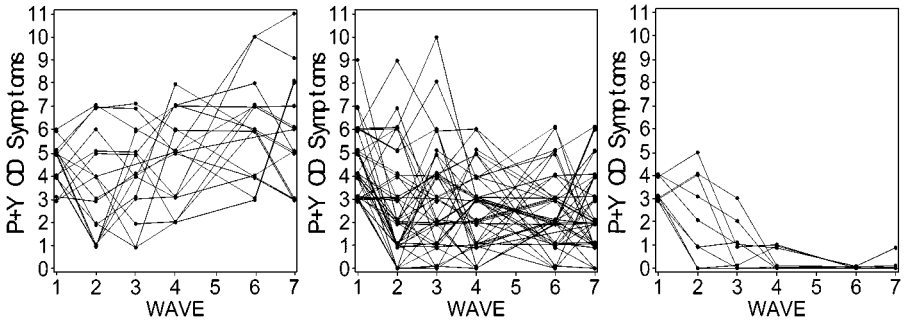

Fig. 2. Individual differences in the outcomes of conduct disorder are shown by plotting the number of conduct disorder behaviors reported by the parent and youth (P + Y) in each wave for each boy when boys who met criteria for conduct disorder in Wave 1 are divided into three arbitrary illustrative groups based on their outcomes defined in terms of their mean numbers of conduct disorder behaviors in Waves 4–7 of ≥4.0 ($n = 17$), 1.0–3.99 ($n = 47$), and <1.0 ($n = 9$).

the range that might be described as sustained recovery from CD behaviors. The predictors and correlates of the broad range of individual differences in the outcomes of CD across Waves 2–7 illustrated in Fig. 2 are the subject of the present paper.

Risk Versus Protective Factors for the Outcomes of CD

Based on the distinction proposed by Stouthamer-Loeber et al. (1993) between risk and protective factors in terms of the shape of the function relating continuous predictor variables to child outcomes, we conducted preliminary analyses of four continuous variables measured in Wave 1 (intelligence, maternal education, total family income, and maternal age when the boy's mother first gave birth to a child) to determine if they should be conceptualized as categorical risk or protective factors for outcomes of childhood CD over the five subsequent assessment waves. Each of the sample distributions of potential predictor scores was divided into their top quartile, middle half, and lower quartile (Fig. 3) and separate planned comparisons among these groups were made using longitudinal log-linear regression analyses for each variable, controlling for age at the start of the study and time. In the case of intelligence, standardization norms were used to divide the range of scores into three levels based on ±1 *SD*.

Because verbal and performance intelligence may be related differently to the persistence of CD (Lynam & Henry, 2001; Moffitt, 1993), they were analyzed separately. Performance intelligence scores did not predict the outcomes of CD over time (across Waves 2–7) when treated as either a continuous or dichotomous variable, but boys with verbal intelligence scores ≥115 ($n = 14$) exhibited fewer CD behaviors over time than boys with verbal intelligence scores of 86–114 ($n = 46$), $\beta = -.48, z = 2.17, p < .03$, and than boys with verbal intelligence scores <86 ($n = 13$), $\beta = -.59, z = -2.34, p < .02$, but boys in the middle half of the distribution of verbal intelligence scores did not exhibit significantly fewer CD behaviors than boys with scores <86, $\beta = -.12, z = -0.77, p = .45$. Using the terminology suggested by Stouthamer-Loeber et al. (1993), then, high verbal intelligence may be viewed as protective against persistent CD. This is because boys with verbal intelligence scores of +1 *SD* or higher have better outcomes of CD than boys at all lower levels of verbal intelligence, but boys at the lowest level of verbal intelligence are no more at risk for poor outcomes of CD than boys in the middle range of verbal intelligence.

Similarly, boys from families in the top quartile of family income in the sample exhibited fewer CD behaviors over time than boys in the middle half of family income, $\beta = -.65, z = -3.42, p < .001$, and than boys in the lower quartile of family income, $\beta = -.52, z = -2.56, p < .02$, but boys in the middle half of the family income did not differ from boys from the lower quartile of income, $\beta = .14, z = 1.08, p = .28$. Again, being from a high income family appears to protect against poor outcomes of CD. Similarly, having a highly educated mother appears to protect against poor CD outcomes. That is, boys in the top quartile of maternal education exhibited fewer CD behaviors over Waves 2–7 than boys in the lower quartile of maternal age, $\beta = -.36, z = -2.04, p < .05$, and than boys in the middle half of the distribution of maternal education, $\beta = -.36, z = -2.07, p < .04$. Boys with mothers in the lower quartile and middle half of maternal education did not differ significantly on CD over waves, however, $\beta = -.01, z = -0.06, p = .95$. These preliminary findings suggest that treating high levels of

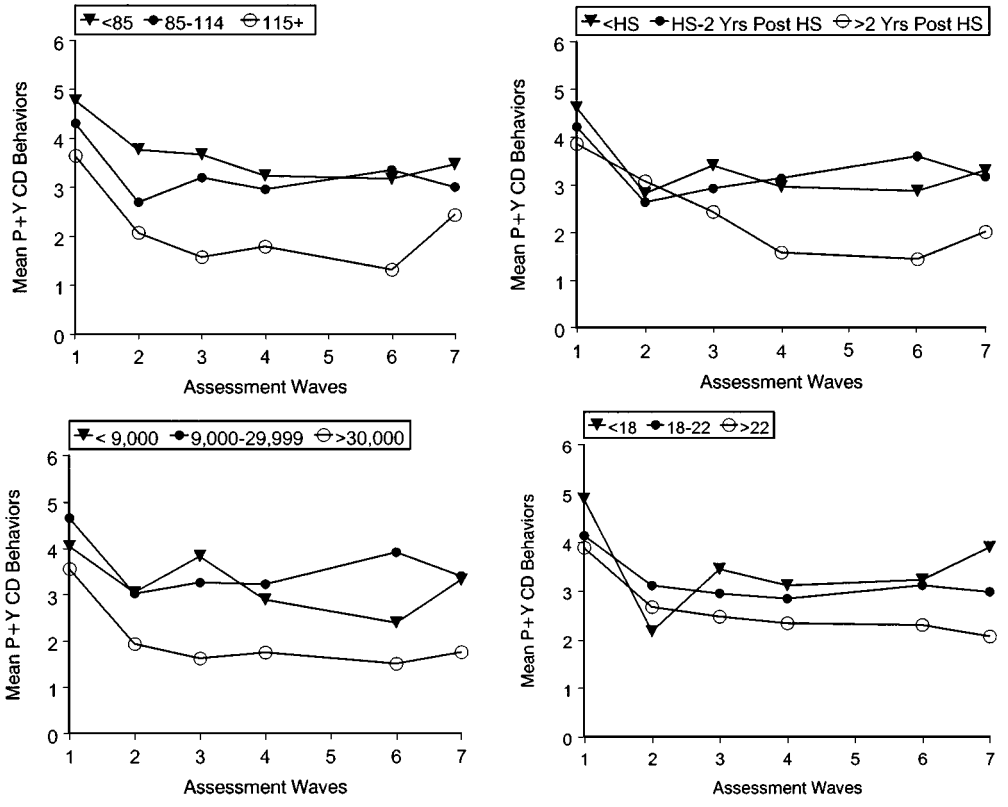

Fig. 3. The mean number of conduct disorder behaviors reported by the parent and youth (P + Y) across Waves 1–7 of boys with verbal intelligence scores of <85, 85–114, or ≥115 (upper left), and who were in the top quartile, middle half, or lower quartile of the sample of maternal education (upper right), total family income (lower left), and maternal age at the birth of her first child.

verbal intelligence, family income, and maternal education as categorical protective factors may maximize the prediction of individual differences in the outcomes of childhood CD.

In contrast, boys in the top quartile of maternal age (older ages at the time of the birth of her first child) exhibited fewer CD behaviors over time than boys in the lower quartile of maternal age, $\beta = -.40, z = 2.54, p < .02$, but not than boys in the middle half of the distribution of maternal age, $\beta = -.26, z = -1.62, p < .10$, and boys in the lower quartile and middle half of maternal did not differ significantly on CD over time, $\beta = -.14, z = -1.11, p = .27$. This suggests that maternal age would be better treated as a continuous predictor of CD outcomes. Because the decision to designate some predictors as protective factors could capitalize on chance differences, however, the findings of the present study must be replicated in other studies to confirm the generalizability of these designations. On the other hand, failing to dichotomize variables that are predictive only at the protective end of their continuous distributions could result in underestimating their prognostic importance.

Baseline Predictors of Individual Differences in Adolescent Outcomes of CD

After determining which predictor variables should be treated as dichotomous protective factors, a comprehensive evaluation was conducted of all variables measured in Wave 1 that might serve as baseline predictors of the subsequent outcomes of CD during Waves 2–7. Controlling for time (waves), the boys' age at the start of the study predicted their level of CD behaviors during Waves 2–7, $\beta = -.09, z = -2.71, p < .01$. Controlling for age at the start of the study and time, individual differences in the course of CD behaviors during Waves 2–7 were also predicted by the number of CD behaviors during Wave 1, $\beta = .15, z = 3.15, p < .005$. The tendency for boys who were older at the start of the study to exhibit fewer CD behaviors across Waves 2–7 did not reflect significantly higher levels of CD behaviors in Wave 1 among younger boys, with the Pearson correlation between age and baseline CD levels being nonsignificant, $r(73) = .13, p = .26$. Age at the start of the study was not found to interact significantly at the $p < .05$ level with either time or the number

Table II. Separate Tests of Potential Baseline (Wave 1) Predictors of Individual Differences in the Outcomes of CD During Waves 2–7, Controlling for Age at the Start of the Study and Time (Waves)

		Not controlling number of CD behaviors in Wave 1			Also controlling number of CD behaviors in Wave 1		
	N	*β*	*z*	*p*	*β*	*z*	*p*
Demographic and socioeconomic factors in Wave 1							
Race–ethnicity (*White* = 0; *African American* = 1)	73	.09	0.71	.48	.08	0.68	.49
Biological parents married/partners (*no* = 0; *yes* = 1)	73	−.17	−1.12	.26	−.10	−0.77	.36
Total family income (top quartile)	70	−.60	−3.25	.001	−.47	−2.57	.01
Maternal education (top quartile)	73	−.13[a]	−1.99	.05	−.13[a]	−2.04	.05
Maternal age at first birth	73	−.02[a]	−2.05	.05	−.02[a]	−2.00	.05
Child characteristics in Wave 1							
WISC-R verbal intelligence (≥115)	73	−.51	−2.33	.01	−.40	−1.93	.05
WISC-R performance intelligence	73	−.00	−0.85	.40	−.00	−0.81	.42
Number of ADHD symptoms	73	.01[a]	1.99	.05	.01[a]	2.00	.05
Number of ODD symptoms	73	.04	1.04	.30	.03	0.90	.37
Number of anxiety symptoms	73	.02	1.05	.29	.01	0.71	.48
Number of depression symptoms	73	.03	1.08	.28	.00	0.02	.98
Maternal psychopathology and substance abuse in Wave 1							
MMPI antisocial index (*T-score* >*60* = 1)	71	.33	2.81	.005	.28	2.53	.01
Substance abuse	73	.43	2.67	.01	.19	0.94	.35
Major depression	73	−.04	−0.29	.77	−.02	−0.12	.90
Paternal psychopathology and substance abuse in Wave 1							
APD	73	.12[a]	2.99	.005	.13[a]	2.99	.005
Substance abuse	73	.10	0.79	.43	.11	0.87	.39
Major depression	73	.03	0.15	.88	.04	0.30	.76

[a] Interaction of predictor with time (assessment waves).

of CD behaviors in Wave 1 in predicting the outcomes of CD.

Other potential baseline predictors of outcome were tested in two sets of analyses. First, each baseline variable was tested in separate analyses as a predictor of individual differences in CD behaviors during Waves 2–7, controlling for age at the start of the study and time (left side of Table II). The number of CD behaviors in Wave 1 was also controlled in the second set of analyses reported in Table II (right side) to examine baseline predictors when the initial severity of CD was controlled. Interactions with time were tested in all cases, but the results were reported only when the interaction term was significant. As shown in Table II, race–ethnicity and the partner status of biological parents in Wave 1 were not significantly related to the adolescent outcomes of CD, but the biological mother's education in Wave 1, total family income in Wave 1, and the mother's age at first birth inversely predicted levels of CD behaviors across Waves 2–7 in both sets of analyses (left and right side of Table II). Boys with higher verbal intelligence scores exhibited fewer CD behaviors in Waves 2–7 in both analyses, but performance intelligence was unrelated to the outcome of CD. The interaction of time with the boys' numbers of ADHD symptoms in Wave 1 was statistically significant. As shown in Fig. 4, boys with higher levels of Wave 1 ADHD symptoms exhibited slightly, but consistently more CD behaviors in Waves 3, 4, 6, and 7, but not in Wave 2.

The boys' numbers of anxiety, depression, and ODD behaviors in Wave 1 did not predict the subsequent outcomes of CD. The latter finding may reflect the restricted range of the number of ODD behaviors in Wave 1 among boys who met criteria for CD, however, with only

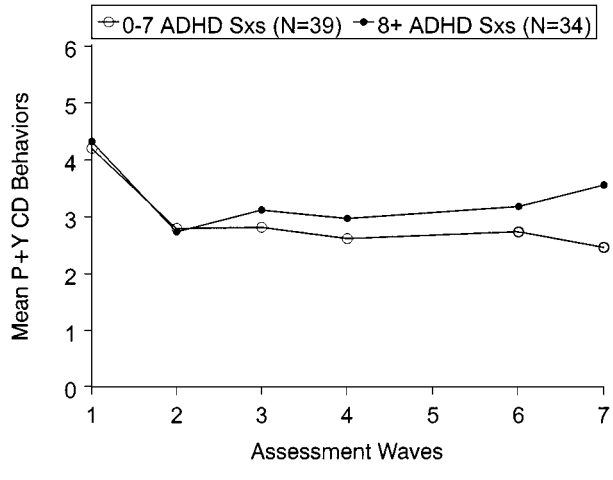

Fig. 4. The mean number of conduct disorder behaviors reported by the parent and youth (P + Y) across Waves 1–7 of boys whose parents reported 0–7 or 8 or more *DSM-III-R* ADHD symptoms (sxs) in Wave 1.

three boys with CD in Wave 1 being reported to have <4 ODD behaviors. That is, ODD behaviors may not have been a predictor of the outcome of CD because high numbers of ODD behaviors were ubiquitous among boys with CD in this sample.

Individual differences in the outcome of CD were also predicted by the mother's MMPI antisocial personality index in both analyses. The diagnosis of APD in the biological father also predicted the boys' outcome in interaction with waves, with the sons of men with APD exhibiting more CD behaviors during later waves than other boys. Maternal substance abuse predicted the outcome of CD, but only when the number of CD behaviors in Wave 1 was not controlled; paternal substance abuse was not related to the boys' CD outcomes in either model.

Joint Models of Baseline Predictors

The baseline predictors of the outcome of CD that were statistically significant when age at the start of the study, time, and the level of CD in Wave 1 were controlled were further examined in a series of joint models. First, the three indicators of socioeconomic status and family demographics (family income, maternal education, and maternal age at first birth) were examined together in a joint model to determine which variables independently predicted the outcome of CD. When the top quartile of maternal education (its interaction with time) and the top quartile of family income were treated as categorical protective factors and maternal age (its interaction with time) was treated as a continuous predictor in log-linear regression, high family income predicted the outcome of CD, $\beta = -.39, z = -2.01, p < .05$, but the maternal education-by-time interaction, $\beta = -.11, z = -1.72, p = .09$, and the maternal age-by-time interaction, $\beta = -.01, z = -1.39, p = .17$, were not significant in the joint model.

It is possible that these three socioeconomic and demographic indicators do not independently predict the outcomes of CD because they are collinear. This was explored in follow-up analyses. Families in the top quartile of family income were found to be more likely to also fall in the top quartile of maternal education than other families (56.2% vs. 9.3%; $OR = 12.6$, 95% CI = 3.3–48.6) and the mother's age at the birth of her first child was found to be older, 19.8 years vs. 18.3 years; $t(68) = -2.91, p < .005$, among families in the top quartile of family income. Therefore, only the top quartile of family income was used to quantify socioeconomic status in all further analyses, as it clearly captured much of the correlated differences in maternal age at first birth and maternal education as well.

Next, a joint model was conducted to determine which aspects of parental psychopathology contributed uniquely to the prediction of the outcomes of CD. Maternal substance abuse was not included in this model because it did not predict the outcomes of CD when the baseline severity of CD was controlled (right side of Table II). Both maternal antisocial personality, $\beta = .28, z = 2.44, p = .02$, and the paternal APD-by-time interactions, $\beta = .13, z = 3.02, p < .005$, were significant, indicating that each baseline variable contributed independently to the prediction of CD outcomes. The three-way interaction of waves and the indicators of maternal and paternal personality was not found to be significant in a subsequent analysis, $\beta = .04, z = 0.81, p = .42$. The additive associations of maternal and paternal APD with their boys' CD behaviors during Waves 2–7 is shown in Fig. 5.

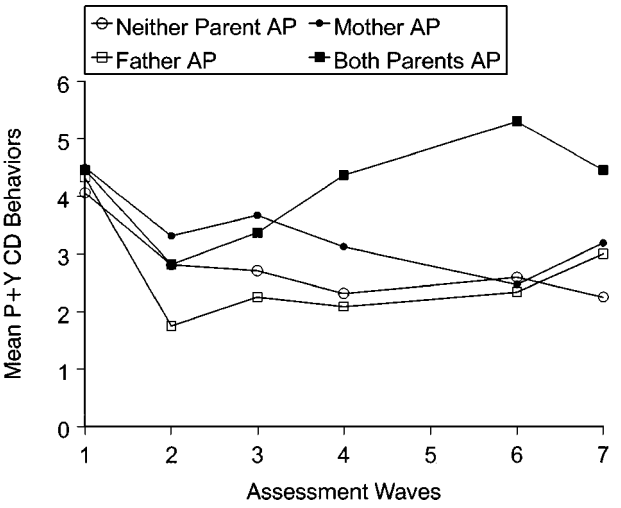

Fig. 5. The mean number of conduct disorder behaviors reported by the parent and youth (P + Y) across Waves 1–7 of boys with no antisocial (AP) biological parents, an antisocial (AP) biological mother, an antisocial (AP) biological father, or two antisocial (AP) biological parents.

Third, a final joint model of baseline variables was conducted that predicted individual differences in the outcomes of CD over Waves 2–7 using time (waves), the boys' age in Wave 1, the boys' number of CD behaviors in Wave 1, the boys' number of ADHD behaviors in Wave 1, being in the top quartile of high family income, maternal antisocial personality, paternal APD, and high verbal intelligence of the child as the predictors. As shown in Table III, the boys' age and baseline number of CD behaviors in Wave 1 each accounted for independent variance in the prediction of outcomes of CD in the final model. In addition, having an antisocial biological mother and/or an antisocial biological father significantly predicted the subsequent outcome of CD, with the strength of the prediction from paternal APD increasing over the five follow-up waves. Three predictors of the outcomes

Table III. Final Joint Model of the Independent Contribution of Each Significant Baseline (Wave 1) Predictor of Individual Differences in the Outcomes of CD Over Waves 2–7

	β	z	p
Time (repeated assessment waves)	−.14	−2.13	.03
Boy's age at start of the study	−.13	−3.86	.0001
Boy's Wave 1 CD behaviors	.12	2.68	.008
Top quartile of family income in Wave 1	−.30	−1.80	.07
Boy's verbal intelligence ≥115	−.30	−1.89	.06
Mother's MMPI antisocial index	.29	2.43	.02
Father's APD-by-time	.13	3.14	.002
Boy's Wave 1 ADHD behaviors-by-time	.06	1.48	.14

Note. APD = antisocial personality disorder. Differences among beta coefficients for predictors reflect both differences in the scale of the predictor and differences in the strength of association. Terms for the father's APD and the boy's Wave 1 ADHD behaviors were included in this model to provide appropriate tests of their interactions with time.

of CD fell to marginal or nonsignificant levels in the final joint model. Living in a family in the top quartile of income and having a verbal intelligence score ≥115 only marginally predicted fewer CD behaviors in Waves 2–7 in this final joint model and the interaction of baseline ADHD-by-time was not significant. Contrary to our earlier report of predictors of the outcome of CD over the first four waves of the present study (Lahey et al., 1995), however, there was not a significant interaction of paternal APD and the child's verbal intelligence in predicting the outcomes of CD across Waves 2–7.

Baseline Prognostic Index

Based on the longitudinal analyses of baseline predictors of the adolescent outcomes of CD, a summary baseline prognostic index was created by summing the following categorical variables measured in Wave 1: (1) >3 CD behaviors in Wave 1; (2) 8 or more ADHD behaviors in Wave 1; (3) verbal intelligence <115; (4) family income in the lower three-quarters of the sample distribution; (5) biological mother with an MMPI antisocial index mean T-score ≥60; and (6) biological father who met diagnostic criteria for APD. The percent of boys with 0 ($n = 1$), 1 ($n = 5$), 2 ($n = 14$), 3 ($n = 21$), 4 ($n = 13$), 5 ($n = 14$), or 6 ($n = 5$) baseline risk variables who exhibited an average of three or more CD behaviors during Waves 4, 6, and 7 were 0, 20, 29, 33, 38, 93, and 100%, respectively. This baseline prognostic index score was trichotomized to facilitate interpretation of its predictive accuracy: A 3-point ordinal prognostic index was defined as 0, 1, or 2 risk factors = 1; 3 or 4 risk factors = 2; and 5 or 6 risk factors = 3. As shown in Fig. 6, log-linear regression

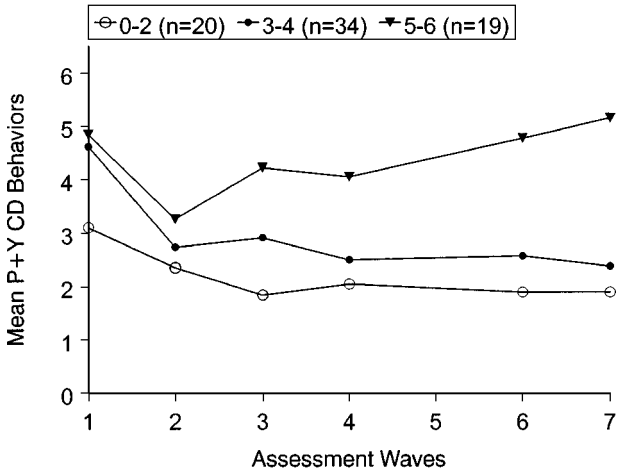

Fig. 6. The mean number of conduct disorder behaviors reported by the parent and youth (P + Y) in Waves 2–7 among boys with scores of 0–2, 3–4, and 5–6 on a summary baseline prognostic index based on significant predictors of the outcomes of CD.

revealed that the 3-point baseline prognostic index significantly predicted the number of CD behaviors during Waves 2–7, $\beta = .36$, $z = 4.40$, $p < .0001$, controlling for time and the boys' age in Wave 1. Furthermore, when the prognostic index-by-time interaction was added to the model, it was significant, $\beta = .07$, $z = 2.10$, $p < .05$, indicating that the 3-point prognostic index may actually predict the future course of CD behaviors somewhat better in waves that are more distant in time from Wave 1. Focusing on these later waves, Table IV shows the odds of exhibiting an average of three or more CD behaviors during Waves 4, 6, and 7 increased 5.8-fold (odds ratio; 95% CI = 2.4–14.1) with each increase of one unit in the 3-point prognostic index, controlling for the boys' age in Wave 1. The accuracy of predicting the outcome of CD defined in this way was substantial when the baseline prognostic index was low (75% true negatives), but especially when it was high (95% true positives).

Table IV. Prediction of an Adverse Outcome of CD, Defined as a Mean of Three or More CD Behaviors During Waves 4, 6, and 7 Using the 3-Point Baseline Prognostic Index Based on Six Baseline Predictor Variables

	Mean of <3 CD behaviors in last three waves	≥3 CD behaviors in last three waves
Baseline prognostic index		
Low	15 (75% true −)	5 (25% false −)
Medium	22	12
High	1 (5% false +)	18 (95% true +)

Time-Varying Covariates

Treatment and Incarceration. Three dichotomous time-varying covariates of receiving psychosocial treatment in the past 12 months, receiving psychoactive medication in the past 12 months, and being placed in juvenile detention or inpatient hospitalization for at least one night in the last 12 months were added to the final joint model of baseline predictors shown in Table III. The level of CD behaviors in each assessment wave was not related to the presence or absence in the same wave of confinement due to either juvenile detention or inpatient hospitalization, $\beta = -.08$, $z = -0.47$, $p = .64$, or the use of outpatient psychotherapy, $\beta = .07$, $z = 0.61$, $p = .54$. The use of psychoactive medication in any given wave was positively associated with higher levels of CD during the same wave, $\beta = .31$, $z = 2.88$, $p < .005$.

Parenting and the Outcomes of CD. We examined the association of parental monitoring and corporal punishment of the boys in Wave t with the number of CD behaviors in Wave $t + 1$, controlling for age and the number of CD behaviors in Wave t. There were no statistically significant associations between wave-to-wave changes in CD and the parents' self-reported use of corporal punishment, $\beta = .10$, $z = 1.38$, $p = .16$, or self-reported parental monitoring, $\beta = -.01$, $z = -0.74$, $p = .46$, in the preceding waves.

When the baseline characteristics of the family that predicted the outcomes of CD (higher family income, maternal antisocial personality, and paternal APD) were included in the model, there were still no statistically significant associations between wave-to-wave changes in CD and the parents' self-reported use of corporal punishment, $\beta = .05$, $z = 0.66$, $p = .51$, or self-reported parental monitoring, $\beta = .00$, $z = 0.22$, $p = .82$.

Because there is evidence that the correlation between physical punishment and child behavior problems is strongest among non-Hispanic White families in the United States (Deater-Deckard, Dodge, Bates, & Pettit, 1996), the analyses of punishment were repeated using only data from the 47 White boys. When the baseline family predictors of the outcomes of CD were not in the model, lagged wave-to-wave changes in CD were significantly associated with corporal punishment in the preceding waves among white boys, $\beta = .23$, $z = 2.41$, $p = .02$. When the family baseline predictors were controlled, however, corporal punishment was not associated with lagged wave-to-wave changes in CD, $\beta = .14$, $z = 1.33$, $p = .18$. There were no significant associations between the course of CD and parental monitoring in either model among the white boys.

DISCUSSION

In the present sample of clinic-referred boys who met diagnostic criteria for CD in Wave 1, most boys continued to engage in significant numbers of CD behaviors from Wave 1 (ages 7–12 years) through the Wave 7 (ages 13–18 years). There were marked individual differences in the course of CD into adolescence, however. All boys showed some degree of fluctuation in symptoms from year to year, but some boys exhibited increasingly more CD behaviors across Waves 2–7, many boys showed little or no decline in CD behaviors over Waves 2–7, and 9 boys improved to a level that might be termed sustained recovery from CD. This suggests that CD is persistent, if fluctuating, for the great majority of prepubertal clinic-referred boys, with less than 15% of such boys showing enough improvement by middle to late adolescence to consider them to be recovered. Therefore, it seems unlikely that previous studies have found that most boys with CD did not meet criteria for APD in adulthood because they recovered from CD during adolescence.

The marked individual differences in the outcomes of childhood CD across Waves 2–7 were predicted by a number of characteristics of the child and family that were measured during baseline (Wave 1). In interpreting the findings from the present analyses, it is important to keep in mind that no adjustment of p-levels was made to protect against Type I errors. Our assumption is that Type II errors (failing to detect associations) is the greater risk in studies using small samples sizes, but it is possible that some of the multiple statistically significant findings of the present analyses reflect chance associations that will not be replicable.

Boys who were older at the start of the study exhibited somewhat fewer CD behaviors across Waves 2–7 and boys exhibiting higher numbers of CD behaviors in Wave 1 showed greater persistence in CD over time. As in previous studies (Lahey et al., 1998; Lynam, 1996), the number of symptoms of ADHD reported by the parent in Wave 1 also predicted the mean number of symptoms of CD in the later follow-up waves to a small, but significant extent. Consistent with the view that verbal aspects of intelligence are more strongly related to CD than nonverbal aspects (Lynam & Henry, 2001; Moffitt, 1993), having a baseline verbal intelligence score of $\geq$115 predicted a more favorable outcome of childhood CD. However, verbal intelligence fell to a marginally significant level ($p = .06$) in the final joint model. This may be because some of the prediction of CD outcomes reflects the association of verbal intelligence with the family sociodemographic indicator also in the model.

A number of correlated indicators of family advantage measured at baseline also contributed to the prediction of future levels of CD. When the child's age at the start of the study and the initial severity of CD were controlled, boys from families with high incomes and high levels of maternal education showed greater improvement in CD behaviors during adolescence, but variations in family income and maternal education within the middle to low range were unrelated to the boys' adolescent outcomes. In contrast, variation across the entire range of maternal age at first birth was inversely related to the adolescent outcome of childhood CD. This confirms two previous longitudinal studies of community samples, which found that children with behavior problems tended to improve from childhood into adolescence if they were from higher socioeconomic status families and had mothers who did not give birth at an early age (Nagin & Tremblay, 2001; Stattin & Trost, 2000). The present study extends previous findings by showing that higher socioeconomic status and older maternal age predict better outcomes of CD even among clinic-referred children who meet criteria for CD during childhood (although these factors were not independent of one another in the present study).

Antisocial personality in the boys' biological mothers (as measured by an antisocial index derived from the MMPI) and the diagnosis of APD in their biological fathers at baseline also combined additively to predict poorer outcomes of CD. In the present study, maternal substance abuse also was a significant predictor of outcome when age at the start of the study was controlled, but not when Wave 1 CD was also controlled. This could also mean that maternal substance abuse is related to the outcomes of CD primarily because it is linked to the initial severity of CD during childhood, which is, in turn, predictive of the long-term course of CD.

The fact that a number of baseline predictors contributed independent variance to the prediction of the outcomes of CD allowed the construction of a summary baseline prognostic index. Although this index will need to be cross-validated in an independent sample to confirm its predictive efficiency, it allowed accurate prediction of the future outcomes of CD behaviors in the present sample (Table III and Fig. 5). This suggests that when the boys in the present study are later assessed for APD in adulthood that the variables in the prognostic index would be good candidates for predictors of which boys with CD during childhood will meet criteria for APD in adulthood, but since most boys showed little consistent improvement in CD during adolescence, it is likely that other factors are involved in predicting which boys with continuing CD in adolescence will meet criteria for APD in adulthood.

The present findings replicate the earlier report by Deater-Deckerd et al. (1996) that parent reports of corporal punishment of the boys in each wave were associated with higher levels of CD in the following wave, controlling for CD in the previous wave, only among non-Hispanic white boys. The association of corporal punishment with the outcomes of CD fell to a nonsignificant level in the present sample when the significant family predictors of the outcomes of CD (higher family income and parental antisocial personality) were added to the model, however. This could indicate that corporal punishment is typical in antisocial families without high economic advantage in non-Hispanic white cultures, but punishment is not itself causally related to the outcomes of CD. On the other hand, one cannot rule out the possibility that the present sample was too small to detect the unique impact of corporal punishment on the outcomes of CD when family characteristics were controlled. Unlike previous studies, parental reports of monitoring were not related to the outcomes of CD, even when family characteristics that predict poor outcomes of CD were not controlled. These findings should be tempered by cautions in interpreting this and other studies that use self-report measures of parenting practices. It is certainly possible that observational or other types of studies might index parenting in more reliable or valid ways that could yield different findings.

Because improvement of CD over time was not positively related to treatment or incarceration over waves, it is important to note that this study was not designed to detect treatment effects. Participants were not randomly assigned to treatments and the treatments were the ones available in the community rather than treatments designed to maximize improvement. Analyses of treatment and incarceration were undertaken only to estimate the extent to which these variables might have affected the course of CD and led to misinterpretation of findings on other predictors. For example, if outpatient psychotherapy had been associated with better outcomes and families with higher incomes were more likely to obtain psychotherapy for their children, it would have been necessary to determine if the association of family income with outcomes of CD was mediated by psychotherapy. Because there was no indication of positive effects of any treatments in this sample, it seems unlikely that treatment distorted the present findings on the predictors of the outcomes of CD. On the other hand, studies that collect more detailed information on treatment and/or are able to randomly assign youths to treatments may find different results.

Taken together, the present findings make good theoretical sense: Boys who meet criteria for CD in childhood are more likely to improve if they have less serious conduct problems and fewer ADHD symptoms at baseline

and have the advantages of high verbal intelligence and affluent and well-educated parents who are not antisocial. Unfortunately, the present study provides little guidance for researchers who study the prevention and treatment of CD. Most of the predictors of the adolescent outcomes of CD identified in this study are not modifiable (i.e., one cannot change the mother's age at the birth of her first child). In addition, the present naturalistic study cannot tell us if intervening to change the theoretically-modifiable predictors would be beneficial. For example, we cannot conclude that helping mothers obtain additional education that would increase family earnings would have a beneficial impact on the adolescent outcomes of their children. This is because it is possible that the family characteristics of children who have poorer adolescent outcome reflect "selection effects" (a tendency for individuals with certain characteristics to select early parenthood, reduced education, and lower-paying career trajectories). The factors underlying such selection effects (such as they parents' own lifelong antisocial tendencies) might prove to be as difficult to modify as their children's antisocial behavior.

ACKNOWLEDGMENT

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REFERENCES

Bardone, A. M., Moffitt, T., Caspi, A., & Dickson, N. (1996). Adult mental health and social outcomes of adolescent girls with depression and conduct disorder. *Development and Psychopathology, 8,* 811–829.

Caspi, A., Taylor, A., Smart, M., Jackson, J., Tagami, S., & Moffitt, T. E. (2001). Can women provide reliable information about their children's fathers? Cross-informant agreement about men's lifetime antisocial behavior. *Journal of Child Psychology and Psychiatry, 42,* 915–920.

Cohen, P., & Brook, J. S. (1995). The reciprocal influence of punishment and child behavior disorder. In J. McCord (Ed.), *Coercion and punishment in long-term perspectives* (pp. 154–164). New York: Cambridge University Press.

Costello, E. J., Edelbrock, C. S., & Costello, A. J. (1985). Validity of the NIMH Diagnostic Interview Schedule for Children: A comparison between psychiatric and pediatric referrals. *Journal of Abnormal Child Psychology, 13,* 579–595.

Costello, A. J., Edelbrock, C., Kalas, R., & Dulcan, M. (1984). *The NIMH Diagnostic Interview Schedule for Children (DISC): Development, reliability, and comparison between clinical and lay interviewers.* Worcester, MA: University of Massachusetts Medical Center.

Deater-Deckard, K., Dodge, K. A., Bates, J. E., & Pettit, G. S. (1996). Physical discipline among African American and European American mothers: Links to children's externalizing behaviors. *Developmental Psychology, 32,* 1065–1072.

Dishion, T. J., & McMahon, R. J. (1998). Parental monitoring and the prevention of child and adolescent problem behavior: A conceptual and empirical formulation. *Clinical Child and Family Psychology Review, 1,* 61–75.

Edelbrock, C., Costello, A. J., Dulcan, M. K., Kalas, R., & Conover, N. C. (1985). Age differences in the reliability of the psychiatric interview of the child. *Child Development, 56,* 265–275.

Edelbrock, C., & Costello, A. J. (1988). Convergence between statistically derived behavior problem syndromes and child psychiatric diagnoses. *Journal of Abnormal Child Psychology, 16,* 219–231.

Eron, L. D., & Huesmann, L. R. (1990). The stability of aggressive behavior—even unto the third generation. In M. M. S. M. Lewis (Ed.), *Handbook of developmental psychopathology. Perspectives in developmental psychology* (pp. 147–156). New York: Plenum.

Esser, G., Schmidt, M. H., & Woerner, W. (1990). Epidemiology and course of psychiatric disorders in school-age children: Results of a longitudinal study. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 31,* 243–263.

Farrington, D. P. (1991). Childhood aggression and adult violence: Early precursors and later-life outcomes. In D. J. Pepler & K. H. Rubin (Eds.), *The development and treatment of childhood aggression* (pp. 5–29). Hilsdale, NJ: Erlbaum.

Farrington, D. P., Loeber, R., & Van Kammen, W. B. (1990). Long-term criminal outcomes of hyperactivity-impulsivity-attention deficit and conduct problems in childhood. In L. N. R. M. Robins (Ed.), *Straight and devious pathways from childhood to adulthood* (pp. 62–81). Cambridge, England: Cambridge University Press.

Fergusson, D. M., Horwood, L. J., & Lynskey, M. T. (1995). The stability of disruptive childhood behavior. *Journal of Abnormal Child Psychology, 23,* 379–396.

Fergusson, D. M., Lynskey, M. T., & Horwood, L. J. (1996). Factors associated with continuity and changes in disruptive behavior patterns between childhood and adolescence. *Journal of Abnormal Child Psychology, 24,* 533–553.

Frick, P. J., & Loney, B. R. (1999). Outcomes of children and adolescents with oppositional defiant disorder and conduct disorder. In H. C. Quay & A. E. Hogan (Eds.), *Handbook of the disruptive behavior disorders* (pp. 507–524). New York: Kluwer Academic/Plenum.

Graham, P., & Rutter, M. (1973). Psychiatric disorder in the young adolescent. *Proceedings of the Royal Society of Medicine, 66,* 1226–1229.

Harrington, R., Fudge, H., Rutter, M., Pickles, A., & Hill, J. (1991). Adult outcomes of childhood and adolescent depression: II. Links with antisocial disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 30,* 434–439.

Hart, E. L., Lahey, B. B., Loeber, R., & Hanson, K. S. (1994). Criterion validity of informants in the diagnosis of disruptive behavior disorders in children: A preliminary study. *Journal of Consulting and Clinical Psychology, 62,* 410–414.

Hathaway, S. R., & Monachesi, E. D. (1952). The Minnesota Multiphasic Personality Inventory in the study of juvenile delinquents. *American Sociological Review, 17,* 704–710.

Hogan, A. E. (1999). Cognitive functioning in children with oppositional and conduct disorder. In H. C. Quay & A. E. Hogan (Eds.), *Handbook of disruptive behavior disorders* (pp. 317–336). New York: Kluwer Academic/Plenum.

Huesmann, L. R., Lefkowitz, M. M., & Eron, L. D. (1978). Sum of MMPI Scales F, 4, and 9 as a measure of aggression. *Journal of Consulting and Clinical Psychology, 46*(5), 1071–1078.

Jensen, P. S., Rubio-Stipec, M., Canino, G., Bird, H. R., Dulcan, M. K., Schwab-Stone, M. E., et al. (1999). Parent and child contributions to diagnosis of mental disorder: Are both informants always necessary? *Journal of the American Academy of Child and Adolescent Psychiatry, 38,* 1569–1579.

Kandel, D. B., & Wu, P. (1995). Disentangling mother–child effects in the development of antisocial behavior. In J. McCord (Ed.), *Coercion and punishment in long-term perspectives* (pp. 106–123). New York: Cambridge University Press.

Kazdin, A. E. (1985). *Treatment of antisocial behavior in children and adolescents.* Homewood, IL: Dorsey Press.

Kilgore, K., Snyder, J., & Lentz, C. (2000). The contribution of parental discipline, parental monitoring, and school risk to early-onset conduct problems in African American boys and girls. *Developmental Psychology, 36,* 835–845.

Lahey, B. B., Loeber, R., Hart, E. L., Frick, P. J., Applegate, B., Zhang, Q., et al. (1995). Four-year longitudinal study of conduct disorder in boys: Patterns and predictors of persistence. *Journal of Abnormal Psychology, 104,* 83–93.

Lahey, B. B., Loeber, R., Quay, H. C., Applegate, B., Shaffer, D., Waldman, I., et al. (1998). Validity of DSM-IV subtypes of conduct disorder based on age of onset. *Journal of the American Academy of Child and Adolescent Psychiatry, 37,* 435–442.

Lahey, B. B., Piacentini, J. C., McBurnett, K., Stone, P. A., Hartdagen, S., & Hynd, G. W. (1988). Psychopathology in the parents of children with conduct disorder and hyperactivity. *Journal of the American Academy of Child and Adolescent Psychiatry, 27,* 163–170.

Lahey, B. B., Russo, M. F., Walker, J. L., & Piacentini, J. C. (1989). Personality characteristics of the mothers of children with disruptive behavior disorders. *Journal of Consulting and Clinical Psychology, 57,* 512–515.

Loeber, R. (1982). The stability of antisocial and delinquent child behavior: A review. *Child Development, 53,* 1431–1446.

Loeber, R. (1991). Antisocial behavior: More enduring than changeable? *Journal of the American Academy of Child and Adolescent Psychiatry, 30,* 393–397.

Loeber, R., Drinkwater, M., Yin, Y., Anderson, S. J., Schmidt, L. C., & Crawford, A. (2000). Stability of family interaction from ages 6 to 18. *Journal of Abnormal Child Psychology, 28,* 353–369.

Loeber, R., Farrington, D. P., Stouthamer-Loeber, M., & Van Kammen, W. B. (1998). *Antisocial behavior and mental health problems: Explanatory factors in childhood and adolescence.* Mahwah, NJ: Erlbaum.

Loeber, R., Green, S. M., Lahey, B. B., Frick, P. J., & McBurnett, K. (2000). Findings on disruptive behavior disorders from the first decade of the Developmental Trends Study. *Clinical Child and Family Psychology Review, 3,* 37–60.

Loeber, R., & Lahey, B. B. (1989). Optimal informants on childhood disruptive behavior disorders. *Development and Psychopathology, 1,* 317–337.

Lynam, D. R. (1996). Early identification of chronic offenders: Who is the fledgling psychopath? *Psychological Bulletin, 120,* 209–234.

Lynam, D. R., & Henry, B. (2001). The role of neuropsychological deficits in conduct disorders. In J. Hill & B. Maughan (Eds.), *Conduct disorders in childhood and adolescence* (pp. 235–263). Cambridge, England: Cambridge University Press.

Masten, A. S., Garmezy, N., Tellegen, A., Pellegrini, D. S., Larkin, K., & Larsen, A. (1988). Competence and stress in school children: The moderating effects of individual and family qualities. *Journal of Child Psychology and Psychiatry, 29,* 745–764.

Moffitt, T. E. (1990). Juvenile delinquency and attention deficit disorder: Boys' developmental trajectories from age 3 to age 15. *Child Development, 61,* 893–910.

Moffitt, T. E. (1993). The neuropsychology of conduct disorder. *Development and Psychopathology, 5,* 135–151.

Moss, H. B., Baron, D. A., Hardie, T. L., & Vanyukov, M. M. (2001). Preadolescent children of substance-dependent fathers with antisocial personality disorder: Psychiatric disorders and problem behaviors. *American Journal on Addictions, 10,* 269–278.

Nagin, D., & Tremblay, R. (2001). Parental and early childhood predictors of persistent physical aggression in boys from kindergarten to high school. *Archives of General Psychiatry, 58,* 389–394.

Offord, D. R., Boyle, M. H., Racine, Y. A., Fleming, J. E., Cadman, D. T., Blum, H. M., Byrne, C., Links, P. S., Lipman, E. L., MacMillan, H. L., Grant, N. I. R., Sanford, N. M., Szatmari, P., Thomas, H., & Woodward, C. A. (1992). Outcome, prognosis, and risk in a longitudinal follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry, 31,* 916–923.

Patterson, G. R., Reid, J. B., & Dishion, T. J. (1998). Antisocial boys. In J. M. O. K. Jenkins (Ed.), *Human emotions: A reader* (pp. 330–336). Malden, MA: Blackwell.

Patterson, G. R., & Stouthamer-Loeber, M. (1984). The correlation of family management practices and delinquency. *Child Development, 55,* 1299–1307.

Pfiffner, L., McBurnett, K., & Rathouz, P. J. (2001). Father absence and familial antisocial characteristics. *Journal of Abnormal Child Psychology, 29,* 357–367.

Piacentini, J. C., Cohen, P., & Cohen, J. (1992). Combining discrepant diagnostic information from multiple sources: Are complex algorithms better than simple ones? *Journal of Abnormal Child Psychology, 20,* 51–63.

Robins, L. (1966). *Deviant children grown up.* Baltimore: Williams and Wilkins.

Robins, L. (1978). Sturdy childhood predictors of adult antisocial behaviour: Replication from longitudinal studies. *Psychological Medicine, 8,* 611–622.

Robins, L., & Ratcliff, K. S. (1979). Risk factors in the continuation of childhood antisocial behavior into adulthood. *International Journal of Mental Health, 7,* 96–115.

Robins, L., West, P. A., & Herjanic, B. L. (1975). Arrests and delinquency in two generations: A study of Black urban families and their children. *Journal of Child Psychology and Psychiatry, 16,* 125–140.

Schonfeld, I. S., Shaffer, D., O'Connor, P., & Portnoy, S. (1988). Conduct disorder and cognitive functioning: Testing three causal hypotheses. *Child Development, 59,* 993–1007.

Spitzer, R., & Endicott, J. (1967). *Schedule for Affective Disorders and Schizophrenia.* New York: New York State Psychiatric Institute.

Spitzer, R., Williams, J. B. W., & Gibbon, M. (1987). *Structured Clinical Interview for DSM-III-R: Non-patient version.* New York: New York State Psychiatric Institute.

Stattin, H., & Magnusson, D. (1989). The role of early aggressive behavior in the frequency, seriousness, and types of later crime. *Journal of Consulting and Clinical Psychology, 57,* 710–718.

Stattin, H., & Trost, K. (2000). When do preschool conduct problems link to future social adjustment problems and when do they not? In L. R. Bergman, R. R. Cairns, L.-G. Nilsson, & L. Nystedt (Eds.), *Developmental science and the holistic approach* (pp. 349–375). Mahwah, NJ: Erlbaum.

Storm-Mathisen, A., & Vaglum, P. (1994). Conduct disorder patients 20 years later: A personal follow-up study. *Acta Psychiatrica Scandinavia, 89,* 416–420.

Stouthamer-Loeber, M., Loeber, R., Farrington, D. P., Zhang, Q., van Kammen, W., & Maguin, E. (1993). The double edge of protective and risk factors for delinquency: Interrelations and developmental patterns. *Development and Psychopathology, 5,* 683–701.

Verhulst, F. C., & Van der Ende, J. (1992). Six-year stability of parent-reported problem behavior in an epidemiological sample. *Journal of Abnormal Child Psychology, 20,* 595–610.

Vuchinich, S., Bank, L., & Patterson, G. R. (1992). Parenting, peers, and the stability of antisocial behavior in preadolescent boys. *Developmental Psychology, 28*(3), 510–521.

Wechsler, D. (1974). *Wechsler Intelligence Scale for Children—Revised.* New York: Psychological Corporation.

Zeger, S. L., & Liang, K.-Y. (1986). Longitudinal data analysis for discrete and continuous outcomes. *Biometrics, 42,* 121–130.

Zoccolillo, M., Pickles, A., Quinton, D., & Rutter, M. (1992). The outcome of childhood conduct disorder: Implications for defining adult personality disorder and conduct disorder. *Psychological Medicine, 22,* 971–986.